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Editor's Choice — Abdominal Compartment Syndrome After Surgery for Abdominal Aortic Aneurysm: A Nationwide Population Based Study[☆]

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WHAT THIS PAPER ADDS

This is the first population based study on abdominal compartment syndrome (ACS) after abdominal aortic aneurysm (AAA) repair, in a contemporary context, namely after the introduction of massive transfusion protocols and the frequent use of endovascular repair (EVAR) after ruptured AAA. ACS was common after ruptured AAA, with similar incidences after open repair (OR) and EVAR, despite the fact that prophylactic open abdomen treatment was frequently used after OR. The clinical consequences of the patient developing ACS were very serious, both in terms of complications and death, emphasizing the importance of developing preventive strategies.

Objective/Background: The understanding of abdominal compartment syndrome (ACS), and its importance for outcome, has increased over time. The aim was to investigate the incidence and clinical consequences of ACS after open (OR) and endovascular repair (EVAR) for ruptured and intact infrarenal abdominal aortic aneurysm (rAAA and iAAA, respectively).
Methods: In 2008, ACS and decompression laparotomy (DL) were introduced as variables in the Swedish vascular registry (Swedvasc), offering an opportunity to study this complication in a prospective, population based design. Operations carried out in the period 2008—13 were analysed. Of 6,612 operations, 1,341 (20.3%) were for rAAA (72.0% OR) and 5,271 (79.7%) for iAAA (41.9% OR). In all, 3,171 (48.0%) were operated on by OR and 3,441 by EVAR. Prophylactic open abdomen (OA) treatment was validated through case records. Cross-matching with the national population registry secured valid mortality data.
Results: After rAAA repair, ACS developed in 6.8% after OR versus 6.9% after EVAR ($p = 1.0$). All major complications were more common after ACS ($p < .001$). Prophylactic OA was performed in 10.7% of patients after OR. For ACS, DL was performed in 77.3% after OR and 84.6% after EVAR ($p = .433$). The 30 day mortality rate was 42.4% with ACS and 23.5% without ACS ($p < .001$); at 1 year it was 50.7% versus 31.8% ($p < .001$). After iAAA repair, ACS developed in 1.6% of patients after OR versus 0.5% after EVAR ($p < .001$). Among those with ACS, DL was performed in 68.6% after OR and in 25.0% after EVAR ($p = .006$). Thirty day mortality was 11.5% with ACS versus 1.8% without it ($p < .001$); at 1 year it was 27.5% versus 6.3% ($p < .001$). When ACS developed, renal failure, multiple organ failure, intestinal ischaemia, and prolonged intensive care were much more frequent ($p < .001$). Morbidity and mortality were similar, regardless of primary surgical technique (OR/EVAR/iAAA/rAAA).
Conclusion: ACS and OA were common after treatment for rAAA. ACS is a devastating complication after surgery for rAAA and iAAA, irrespective of operative technique, emphasizing the importance of prevention.

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INTRODUCTION

Abdominal compartment syndrome (ACS) is a serious complication after surgery for abdominal aortic aneurysm (AAA) and it is more common after rupture (rAAA).[1] If left untreated, mortality is nearly 100%; with treatment, mortality is still 30—70%.[2—4] Although the focus in recent years has been on prevention, early recognition is decisive for outcome, and definitive treatment with open abdomen (OA) is often required, where the treatment itself is also a

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cause of morbidity.[5,6] Routine measurement of intra-abdominal pressure (IAP) in the critically ill is an established routine in many intensive care units.[6]

The incidence of ACS requiring treatment with OA is low after repair of intact AAA (iAAA), 0.4% after endovascular repair (EVAR) and 0.9% after open repair (OR).[4] The corresponding risks after repair of rAAA depend on case mix, the proportion of patients treated prophylactically with OA, and the resuscitation routines.[7] After OR for rAAA, approximately 20% of patients will suffer from ACS,[8–10] and treatment with OA will be required in 2.5–7.4% of the cases.[4,8] In a recent meta-analysis, 8–17% of patients treated for rAAA with EVAR developed ACS.[11] In earlier Swedish studies, 3.4–6.9% of patients required treatment with OA after EVAR for rAAA.[4,12] In a Swiss study,[2] 20% of patients treated with EVAR for rAAA required treatment with OA, in an experience when virtually all patients with rAAA were treated with EVAR.

The prevalence of AAA is declining,[13–15] yet at the same time the number of repairs is increasing,[16] which is explained by screening and the introduction of EVAR, enabling elective surgery on older and frailer patients. The proportion of patients treated with EVAR for iAAA has surpassed 50% in many countries, and a continued increase is also seen for patients with rAAA.[16,17] Peri-operative care has improved in general,[18] and the introduction of massive transfusion protocols has the potential to decrease the incidence of ACS.[6,19,20] Massive transfusion protocols aim at securing the logistics of haemostatic resuscitation, with transfusing fresh frozen plasma, packed red blood cells, and platelets at a ratio of 1:1:1,[21] while limiting the volume of crystalloid solutions.[22]

With this changing landscape, it would be logical to assume that the incidence of ACS will be affected. The aim of this study is to describe the contemporary incidence, treatment, and outcome of ACS after AAA repair in Sweden.

METHODS

Following the first international consensus conference on ACS in 2004, the Definitions and Recommendations were published in 2006 and 2007.[23,24] Since May 2008, ACS and decompression laparotomy (DL) are registered prospectively in the nationwide vascular registry, Swedvasc. All infra- and juxta-renal AAA repairs registered at the 32 Swedish hospitals performing aortic surgery between May 2008 and December 2013 were identified. Entering the patient into this module of the registry was entirely based on the anatomy of the reconstruction. From this cohort, patients with ACS and DL were further analysed.

Duplicate entries, elective endovascular re-do procedures for leakage, repairs for diagnoses other than AAA, on table deaths, and incomplete or faulty registrations were excluded. Correcting the database is facilitated by the fact that every Swedish citizen and permanent resident has a unique 10 digit personal identity number. Patients who had EVAR converted to open surgery were excluded from the analyses comparing OR and EVAR. The university hospital in Gothenburg was excluded on the basis of an ongoing study leaving all abdomens open after repair of rAAA.

Swedvasc has previously been validated in a number of studies,[25–28] showing national coverage (all hospitals in Sweden performing elective AAA repair report to the registry) and external validity well above 90%. In a recent validation,[29] performed by international independent experts, the external validity was 99.5%, with only two missing cases of 417 AAA repairs performed at five randomly selected hospitals in 2012. The national population registry is cross-linked with Swedvasc, updating the database with new dates of death each week, ensuring near complete accuracy regarding survival data. Patients operated on within 1 year from the data extraction in November 2014, and thereby not completing one whole year of follow up, were excluded from evaluation of 1 year mortality, but included in all other analyses.

ACS was defined according to the consensus document and clinical practice Guidelines of the Abdominal Compartment Society:[18,23] a sustained intra-abdominal pressure >20 mmHg that is associated with new organ dysfunction/failure.

Data were analysed separately for patients treated for intact and ruptured AAA, as well as for OR and EVAR. Pre-operative comorbidities included diabetes, cardiac risk (previous or current heart disease or cardiac intervention), cerebrovascular disease, pulmonary disease, and renal insufficiency (defined as a serum creatinine >150 μmol/L).

Validation was performed in the rAAA group to identify the prevalence of leaving the abdomen open at the end of a primary OR. Case records of 300 randomly selected patients operated on for ruptured AAA were requested from the respective hospitals.

Statistical analysis

Fisher's exact test was used for comparison of proportions. Independent samples *t* test was used for continuous variables after testing for normally distributed data. All tests were two sided and a p value $< .01$ was considered statistically significant, adjusting for multiple comparisons. Long-term survival was analysed with Cox regression. Statistical evaluation was performed with SPSS Statistics version 22 (IBM, Armonk, NY, USA).

Ethical considerations

The regional ethical review board in Uppsala approved the study.

RESULTS

During the study period (May 2008–December 2013), 7,414 registrations from 31 separate institutions were identified, of which 6,634 patients were included. The cohort, with exclusions is summarized in Fig. 1.

There were 5,271 repairs for iAAA and 1,341 repairs for rAAA (20.4%). When treated for iAAA, 2,206 received OR (41.9%) and when treated for rAAA, 965 received OR

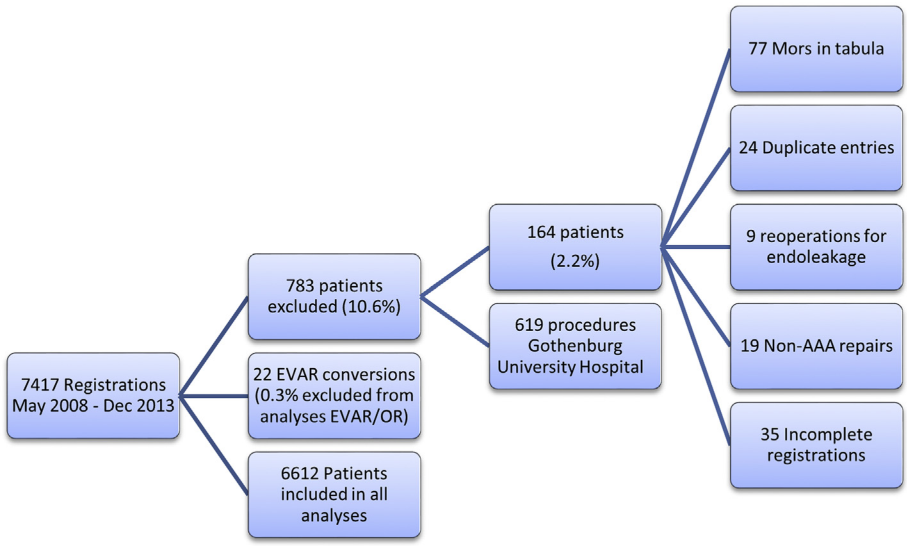

Figure 1. Study design. *Note.* EVAR = endovascular aneurysm repair; OR = open repair; AAA = abdominal aortic aneurysm.

(72.0%). The characteristics of the cohort are presented in Table 1.

In the rAAA group, 66 patients (6.8%) treated with OR developed ACS versus 26 (6.9%) treated with EVAR ($p = 1.0$), among whom DL was performed in 51 (77.3%) after OR versus 22 (84.6%) after EVAR ($p = .433$).

In the iAAA group, 35 patients (1.6%) developed ACS after OR versus 16 (0.5%) after EVAR ($p < .001$), among whom DL was performed in 24 (68.6%) after OR versus four (25%) after EVAR ($p = .004$).

Risk factors for ACS

The risk factors for ACS are described in Table 2. Among the pre-operative comorbidities, none was identified as a significant risk factor for ACS.

Within the rAAA group, ACS was associated with the lowest measured pre-operative blood pressure and pre-operative unconsciousness.

ACS was more common in both the iAAA and rAAA groups after peri-operative bleeding of >5 L, in the iAAA group after re-implantation of a renal artery, and in the rAAA group after the use of balloon occlusion during EVAR. Among those operated on for iAAA the risk of developing ACS was 8.1% among those who suffered peri-operative bleeding >5 L compared with only 0.8% if the bleeding was <5 L ($p < .001$).

Outcome with ACS

The outcome of patients with ACS was significantly worse than their counterparts after both rAAA and iAAA repairs. As noted in Table 3, among patients treated for rAAA, acute myocardial infarction (AMI), multi-organ failure (MOF), renal failure, intestinal ischaemia, and bowel resection were all significantly more common with ACS (all $p < .001$). Among patients treated for iAAA, MOF, renal failure, intestinal ischaemia, and bowel resection were more common (all $p < .001$), while there was a trend for AMI ($p = .05$). In the rAAA group, the 30 day mortality rate with ACS was 42.4%, rising to 58.7% at 90 days and 60.7% at 1 year. Without ACS, the 30 day mortality rate was 23.5%, rising to 27.2% at 90 days and 31.8% at 1 year ($p < .001$).

After iAAA repair, the 30 day mortality rate with ACS was 11.5%, rising to 19.2% at 90 days and 27.5% at 1 year. Without ACS the corresponding mortality rates were 1.8%,

Table 1. Clinical characteristics of the Swedvasc cohort.

	All patients	Ruptured AAA			Intact AAA		
	(n = 6,612)	OR (n = 965)	EVAR (n = 376)	p[a]	OR (n = 2,206)	EVAR (n = 3,065)	p[a]
Age (y)	72.8	73.9	76.6	**<.001**	69.9	74.0	**<.001**
Female sex (%)	16.6	17.6	23.1	.025	17.6	14.9	**.009**
Cardiac disease (%)	40.0	36.5	42.6	.067	36.7	43.7	**<.001**
Pulmonary disease (%)	22.9	22.6	26.7	.164	21.6	23.5	.120
Creatinine (µmol/L)	99.0	123.3	108.3	.191	90.2	98.6	**<.001**
Mean aortic diameter (mm)	64.0	79.4	73.1	.218	61.4	61.0	.400
Peri-operative bleeding > 5 L (%)	6.8	31.1	3.7	**<.001**	5.7	0.2	**<.001**
ACS (%)	2.2	6.8	6.9	1.0	1.6	0.5	**<.001**
DL (%)	71.2	77.3	84.6	.433	68.6	25	**.006**

Note. Data in bold denote significant values. AAA = abdominal aortic aneurysm; OR = open repair, EVAR = endovascular aneurysm repair; ACS = abdominal compartment syndrome; DL = decompression laparotomy among those who developed ACS.

[a] Refers to comparison between OR and EVAR.

Table 2. Risk factors for abdominal compartment syndrome (ACS).

	Ruptured AAA ACS (n = 94)	No ACS (n = 1,253)	p[a]	Intact AAA ACS (n = 52)	No ACS (n = 5,235)	p[a]
Age (y)	74.1	74.7	.466	72.0	72.3	.758
Female sex (%)	18.1	19.1	.892	17.3	16.0	.849
Diabetes (%)	10.2	12.5	.617	17.0	12.7	.377
Cardiac disease (%)	30.9	38.5	.192	42.6	40.5	.768
Cerebrovascular disease (%)	7.6	15.7	.052	17.0	13.3	.395
Pulmonary disease (%)	26.3	23.6	.587	25.5	22.7	.603
Creatinine (μmol/L)	118.9	117.0	.797	95.8	95.1	.914
Mean aortic diameter (mm)	77.4	76.6	.823	60.1	61.2	.675
Lowest blood pressure (mmHg)	61.4	76.3	**.004**	–	–	–
Unconsciousness pre-operatively (%)	60.4	44.9	**.004**	–	–	–
Peri-operative bleeding > 5 L (%)	44.7	22.1	**<.001**	21.2	2.4	**<.001**
Aorto-bi-femoral reconstruction (%)	13.2	8.0	.167	19.4	8.5	.032
Re-implantation of renal artery (%)	4.4	1.1	.057	13.9	3.5	**.009**
Aortic balloon occlusion (%)	61.5	20.2	**<.001**	6.3	1.4	.212

Note. Data in bold denote significant values. AAA = abdominal aortic aneurysm.
[a] Refers to comparison between ACS and no ACS.

3.0%, and 6.3% ($p < .001$). The cumulative survival is presented in Fig. 2 (rAAA) and Fig. 3 (iAAA). Although the number of patients at risk decreases over time owing to a high mortality, 100% of the included patients had information on survival through cross-linking with the population registry.

There were no differences in mortality among patients who developed ACS, depending on whether they underwent DL or not (Table 4).

Outcome with ACS depending on surgical method

In the rAAA group, patients with ACS after EVAR were older, with a mean age of 77.3 years compared with 72.8 years after OR ($p = .007$; Table 5). When ACS developed after iAAA repair, age and sex did not differ between those treated by EVAR or OR.

In the iAAA group there was a difference in the frequency of intensive care unit stay >5 days, with 74.3% after OR versus 31.3% after EVAR ($p = .005$). There was a trend towards increased MOF after OR (42.9% vs. 12.5%; $p = .057$).

There was no difference in mortality between EVAR and OR among those who developed ACS. After rAAA and OR the mortality rate was 37.5% at 30 days, 54.7% at 90 days, and 54.0% at 1 year. The corresponding mortality rates after EVAR were 50% at 30 days ($p = .346$), 65.4% at 90 days ($p = .481$), and 75.0% at 1 year ($p = .09$). In a Cox

Table 3. Outcomes depending on abdominal compartment syndrome (ACS) or not.

	Ruptured AAA ACS (n = 94)	No ACS (n = 1,253)	p[a]	Intact AAA ACS (n = 52)	No ACS (n = 5,235)	p[a]
Age (y)	74.1	74.7	.466	72.0	72.3	.758
Female sex (%)	18.1	19.2	.892	17.3	16.0	.849
AMI (%)	14.6	4.4	**<.001**	5.9	1.6	.05
Renal failure (%)	73.1	15.6	**<.001**	48.1	3.5	**<.001**
MOF (%)	63.4	11.5	**<.001**	34.6	1.0	**<.001**
ICU care > 5 d (%)	97.4	22.7	**<.001**	61.5	3.4	**<.001**
Intestinal ischaemia (%)	38.5	7.1	**<.001**	28.8	1.2	**<.001**
Intestinal resection (%)	28.7	3.6	**<.001**	25.0	0.7	**<.001**
Re-lap for bleeding (%)	28.7	5.0	**<.001**	19.2	2.2	**<.001**
Death < 30 d (%)	42.4	23.5	**<.001**	11.5	1.8	**<.001**
Death < 90 d (%)	58.7	27.2	**<.001**	19.2	3.0	**<.001**
Death < 1 y (%)	60.7	31.8	**<.001**	27.5	6.3	**<.001**

Note. Data in bold denote significant values. AAA = abdominal aortic aneurysm; AMI = acute myocardial infarction; MOF = multi-organ failure; ICU = intensive care unit.
[a] Refers to comparison between ACS and no ACS.

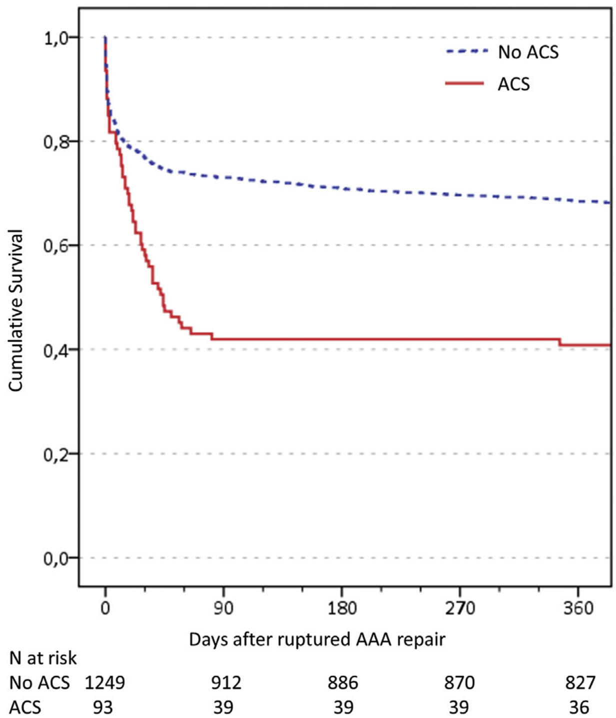

Figure 2. Survival after ruptured abdominal aortic aneurysm (AAA) repair with or without abdominal compartment syndrome (ACS).

regression model, age (p = .153), sex (p = .411), and operative technique (p = .218) did not influence survival.

In the iAAA group, the mortality rate after OR was 14.3% at 30 days, 20.0% at 90 days, and 20.6% at 1 year. After EVAR, the mortality rate was 6.3% at 30 days (p = .651), 18.8% at 90

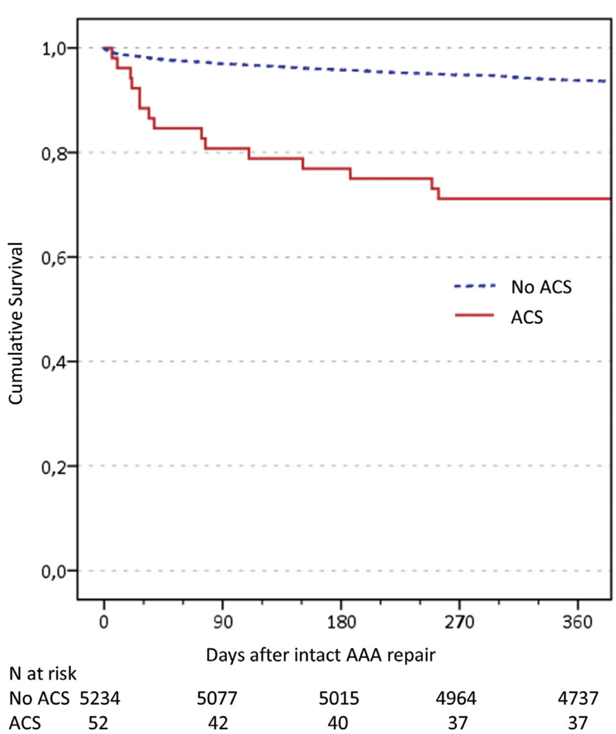

Figure 3. Survival after intact abdominal aortic aneurysm (AAA) repair with or without abdominal compartment syndrome (ACS).

days (p = 1.0), and 43.8% at 1 year (p = .105). In a cox regression model, age (p = .097), sex (p = .227), and operative technique did not influence mortality (p = .496).

Treatment with open abdomen

Of the 1,347 patients in the rAAA group, 300 of 965 treated with OR (31.1%) were randomly selected for validation regarding prophylactic treatment with OA, regardless of whether ACS was present or not. Information from case records in 289 of those (96.3%) was obtained. The abdomen was left open in 31 patients at the primary operation (10.7%; 95% confidence interval [CI] 7.2—14.3). Four of those patients (4/289; 1.4%) had been erroneously registered as having had ACS (which they never developed, as they had prophylactic OA, but there was no way of recording this event in the registry), and they were also registered as having had DL. In the rAAA group as a whole, 75/1,347 (5.6%) patients were registered as having undergone DL in relation to ACS; however, if the erroneously registered 1.4% in the validated cohort is subtracted, this estimation is reduced to 4.2%. Thus, in all, approximately 14.9% (10.7% + 4.2%) required OA treatment after rAAA repair. In the validated cohort, 15 of the 258 patients who had their abdomen closed developed ACS (5.8%), and of those 14 (93%) underwent DL.

After iAAA repair 29 patients were treated with DL (0.5%). With such a low frequency a proper validation of the patients treated for iAAA was not considered feasible.

DISCUSSION

Leaving the abdomen open at the primary laparotomy, as well as after DL because of later ACS, were both found to be common after rAAA repair. Approximately 15% were treated with OA after OR of a rAAA. The majority with ACS underwent DL, with the exception after EVAR for iAAA, when a surprisingly low proportion of the patients underwent laparotomy. In both the iAAA and rAAA groups, outcomes with ACS were significantly worse in nearly all aspects of morbidity and mortality, but there were, in general, no differences in outcome, dependent on the primary operative technique. Thus, ACS is a devastating complication after rAAA repair, regardless of whether an open or endovascular technique is used. This knowledge is particularly important given the fact that ACS developed as often after EVAR as after OR (although prophylactic OA treatment was common after OR).

In contrast, the incidence of ACS after iAAA repair was low, and a significant difference was seen between OR and EVAR (1.6 vs. 0.5%; $p < .001$). Comparisons with other studies are difficult, as ACS after iAAA has not been widely reported, and the present study is, to the authors' knowledge, the first large population based study. In a recent study reporting from four Swedish hospitals with an interest in this clinical issue, and thus probably with a higher detection rate, five of 758 (0.7%) were treated with OA after iAAA repair.[4] That figure can be compared with the proportion treated with DL in the present study (28/5,271; 0.5%). Among those developing ACS in the iAAA group, the

Table 4. Outcome after abdominal compartment syndrome (ACS) if decompression laparotomy (DL) carried out or not.

	Ruptured AAA with ACS			Intact AAA with ACS		
	DL (n = 74)	No DL (n = 18)	p[a]	DL (n = 29)	No DL (n = 23)	p[a]
Death < 30 d	29 (39.2)	10 (55.6)	.288	4 (13.8)	2 (8.7)	.682
Death < 90 d	44 (59.5)	10 (55.6)	.794	7 (24.1)	3 (13.0)	.482
Death < 1 y	45 (60.8)	10 (55.6)	.790	10 (34.5)	5 (21.7)	.369

Note. Data are n (%) unless otherwise indicated. AAA = abdominal aortic aneurysm.

[a] Refers to comparison between patients undergoing DL or not (no DL).

proportion decompressed after OR was slightly lower than those in the rAAA group, but after EVAR the decompression rate was only 25%. This was an unexpected finding, which raises questions about the intra-abdominal pressures recorded, their duration, and how the patients were otherwise treated. Despite the fact that the responsible clinicians thought there was no need for DL in 75% of cases, these patients had poor survival, with a mortality rate of 43.8% versus 6.0% without ACS at 1 year ($p < .001$), suggesting that the threshold for DL should possibly have been lower. The immediate effects of DL on IAP, as well as on renal and pulmonary function, were recently demonstrated in a multicentre study.[30]

The incidence of ACS in the rAAA group was 6.8% after OR and 6.9% after EVAR, compared with 8—20% in previous studies.[2,11] There are multiple confounding factors, among them case mix and how often prophylactic treatment with OA was used in the OR group, precluding far reaching conclusions, but the fact that there was no difference is thought provoking. Even though patients treated with OR were haemodynamically unstable more often, with lower pre-operative blood pressures and higher rates of unconsciousness (both $p < .001$), 10.7% were also prophylactically treated with OA. As most centres use prophylactic treatment with OA more or less selectively, illustrated by both similar and higher rates in previous publications,[20,31] the optimal strategy has yet to be determined.

After rAAA repair, treatment of ACS required DL in 75/1,347 (5.6%). Adding those treated prophylactically with OA after primary OR, the rate of OA treatment was 14.9% in this subgroup. Recent literature reports both a higher and lower rate with OA,[2,4] where the differences in this context can also be explained by both confounding factors and differences in clinical practice. A shift from recognition and treatment towards prevention of ACS has taken place globally,[18,19,32,33] and to what extent this intellectual process has taken place among the responsible vascular surgeons and intensivists will affect clinical practice.

The morbidity and mortality rates associated with ACS were devastating both in the iAAA and rAAA groups. Mortality in surgical studies is generally reported at 30 days, which in previous reports on rAAA repair were 30—70%,[2,3,11] and in this study it was 42.4%. It is desirable, however, and more appropriate to report on the mortality at 90 days. It is evident in this study, and especially in the rAAA group, that mortality stabilized first after 90 days. In this group, the mortality increased to 58.7% at 90 days, twice the rate without ACS. The corresponding rate for those with ACS after iAAA repair was 19.2%, a figure six times higher than that without ACS. When comparing OR with EVAR, there were few significant differences and none regarding mortality. Despite modern intensive care and increasing use of minimal invasive surgery (EVAR),[16] outcome is truly devastating. For the clinician, these

Table 5. Outcome after abdominal compartment syndrome (ACS) if open (OR) or endovascular repair (EVAR).

	Ruptured AAA with ACS			Intact AAA with ACS		
	OR (n = 66)	EVAR (n = 26)	p[a]	OR (n = 35)	EVAR (n = 16)	p[a]
Age (y)	72.8	77.3	**.007**	71.1	73.8	.139
Aneurysm diameter (mm)	79.4	73.1	.218	61.9	55.8	.168
Female sex (%)	13.6	30.8	.075	11.4	31.3	.118
AMI (%)	15.9	12.5	1.0	5.9	6.3	1.0
Renal failure (%)	77.3	64.0	.20	51.4	37.5	.384
MOF (%)	65.2	56.0	.471	42.9	12.5	.053
ICU care > 5 days (%)	96.5	100	1.0	74.3	31.3	.005
Intestinal ischaemia (%)	40.6	32.0	.479	22.9	43.8	.187
Intestinal resection (%)	30.3	23.1	.610	17.1	43.8	.08
Re-lap for bleeding (%)	25.8	34.6	.445	20.0	18.8	1.0
DL (%)	77.3	84.6	.572	68.6	25.0	**.006**
Death < 30 d (%)	37.5	50.0	.346	14.3	6.3	.651
Death < 90 d (%)	54.7	65.4	.481	20.0	18.8	1.0
Death < 1 y (%)	54.0	75.0	.090	20.6	43.8	.105

Note. Patients who were converted from EVAR to OR (n = 3) were excluded from this analysis. Data in bold denote significant values. AAA = abdominal aortic aneurysm; AMI = acute myocardial infarction; MOF = multi-organ failure; ICU = intensive care unit; DL = decompressive laparotomy.

[a] Refers to comparison between ACS and no ACS.

figures highlight the need for a high index of suspicion in every patient at risk, ensuring optimal prevention and treatment of ACS.

Age, sex, and pre-operative comorbidities were not associated with ACS. Parameters indicating a larger blood loss in patients with rAAA and a more complex surgical procedure were identified as risk factors, consistent with earlier findings.[3] In the Swedvasc registry aortic diameter is noted as the sole variable describing aortic morphology. The IMPROVE trial recently reported short neck length to have a strong adverse influence on mortality after surgery for rAAA.[34] It has been decided to add this variable to the Swedvasc registry, making future analyses possible.

With such poor results among patients who developed ACS, prevention is the obvious key to success. Massive transfusion protocols and permissive hypotension in patients with ongoing bleeding are important, as well as being restrictive with crystalloids.[6,7] A proactive strategy treating intra-abdominal hypertension with medical therapy such as effective pain relief and neuromuscular blockade are other important preventive measures.[19,20] This study has limitations. Data were entered prospectively into the Swedvasc registry but by many different vascular surgeons that may have interpreted the definition of ACS in different ways. However, there is a help text in the registry, giving the consensus definition from the World Society of the ACS,[6,23] and the issue of ACS has been intensely discussed at meetings during the last two decades. As noted in the validation, there were some erroneous registrations confounding the prophylactic use of OA with ACS and DL, but this error was investigated and could be estimated. External validity of the registry has recently been tested by an international independent audit committee, and was found to be 99.5% during 2012.[29] Thus, there are very few missing cases. The exclusion of one major hospital, owing to a competing research project, would probably not introduce any bias to the study, and other exclusions constituted only 2.2% of the entire cohort (see Fig. 1).

It is important to emphasize that the 6,634 patients studied all had infrarenal AAA repair. Patients with thoraco-abdominal, supra-, or juxta-renal aortic aneurysms may have different risks of developing ACS. Patients with juxta-renal AAA should be included in the present study, but the distinction between juxta- and supra-renal AAA is not always clear, in particular when endovascular techniques are used.

When comparing morbidity and mortality between EVAR and OR among those who developed ACS in the iAAA group, many of the analyses show quite large numerical differences, which still are insignificant. Even though the iAAA group consisted of 5,271 patients and is the largest study to date, it may still yield too few cases with ACS to demonstrate associations, resulting in type II statistical error.

Conclusion

In this first ever large population based study, both ACS and leaving the abdomen open at the primary operation were common after treatment for rAAA. ACS was as common after EVAR as after OR in this setting, despite the fact that prophylactic OA treatment was common after OR. ACS is associated with a devastating effect on outcome after surgery for both ruptured and intact AAA. There was no difference in outcome among those who developed ACS, depending on whether the primary treatment had been performed with an open or endovascular technique.

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CONFLICT OF INTEREST

None declared.

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